dependent on culture, although this point requires evaluation with a larger number of clinical specimens. Such large-scale studies on clinical specimens will also be required before this procedure might be of interest for the diagnosis of mycobacterial infection. In particular, decontamination of clinical specimens and contamination of cultures by non-mycobacterial strains might influence the sensitivity and specificity of the method and require careful evaluation.

References

1. Wilton S, Cousins D. Detection and identification of multiple mycobacterial pathogens by DNA amplification in a single tube. PCR Methods Applicat 1992; 1: 269–73.
2. Clarridge JE, Shawar RM, Shinik TM, Plikaytis BB. Large-scale use of polymerase chain reaction of *Mycobacterium tuberculosis* in routine mycobacteriology laboratory. J Clin Microbiol 1993; 31: 2049–56.
3. Forbes BA, Hicks KES. Direct detection of *Mycobacterium tuberculosis* in respiratory specimens in a clinical laboratory by polymerase chain reaction. J Clin Microbiol 1993; 31: 1688–94.
4. Wobeser WL, Krajden M, Conly J, et al. Evaluation of Roche Amplicor PCR Assay for *Mycobacterium tuberculosis*. J Clin Microbiol 1996; 34: 134–9.
5. Del Portillo P, Thomas MC, Martinez E, et al. Multiprimer PCR System for differential identification of mycobacteria in clinical samples. J Clin Microbiol 1996; 34: 324–8.
6. Somoskövi A, Magyar P, Szilàgyi ZS, Muràszki K, Gulyàs K. Comparison of MB/Redox vs. MGIT, Middlebrook 7H11 and Löwenstein–Jensen media for isolation of mycobacteria in clinical specimens. Quimioterapia 1997; 10(suppl 2): 93.
7. Nolte FS, Metchock B. *Mycobacterium*. In Murray PR, Baron EJ, Pfaller MA, Tenover FC, Yolken RH, eds. Manual of clinical microbiology, 6th edn. Washington, DC: American Society for Microbiology, 1995: 400.
8. LaBombardi VJ, Cataldo-Caputzal L. Ciprofloxacin susceptibility testing by MIC and disk elution of drug-resistant *Mycobacterium tuberculosis* and *Mycobacterium avium* complex. Antimicrob Agents Chemother 1993; 37: 1556–7.
9. Shawar RM, El-Zaatari FAK, Nataraj A, Clarridge JE. Detection of *Mycobacterium tuberculosis* in clinical samples by two-step polymerase chain reaction and nonisotopic hybridization methods. J Clin Microbiol 1993; 31: 61–5.
10. Kulski JK, Khinsoe C, Pryce T, and Christiansen K. Use of multiplex PCR to detect and identify *Mycobacterium avium* and *M. intracellulare* in blood culture fluids of AIDS patients. J Clin Microbiol 1995; 33: 668–74.

Rapidly progressive tricuspid valve endocarditis caused by *Capnocytophaga canimorsus* infection in an immunocompetent host

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Capnocytophaga canimorsus is a Gram-negative, fusiform, micro-aerophilic rod. The organism is part of the normal oral flora in cats and dogs [1]. The estimated incidence of septicemia in humans is 0.5 cases per million inhabitants per year [2]. The disease especially affects asplenic or alcoholic individuals and patients using steroids. Fifty per cent of the patients had been bitten by a dog in the week before admission [2].

C. canimorsus has been identified as a cause of tricuspid valve endocarditis in patients with risk factors for infection with *C. canimorsus* or with cardiac abnormalities. We report a case of rapidly progressive right ventricular failure due to right-sided endocarditis caused by *C. canimorsus* in an immunocompetent woman without structural cardiac defects.

A 69-year-old woman with a history of chronic obstructive pulmonary disease (COPD) developed progressive edema in both legs in 2 weeks, without progressive dyspnea or orthopnea. She had no history of cardiac disease. On examination she was slightly dyspneic, with a blood pressure of 110/85 mmHg, a

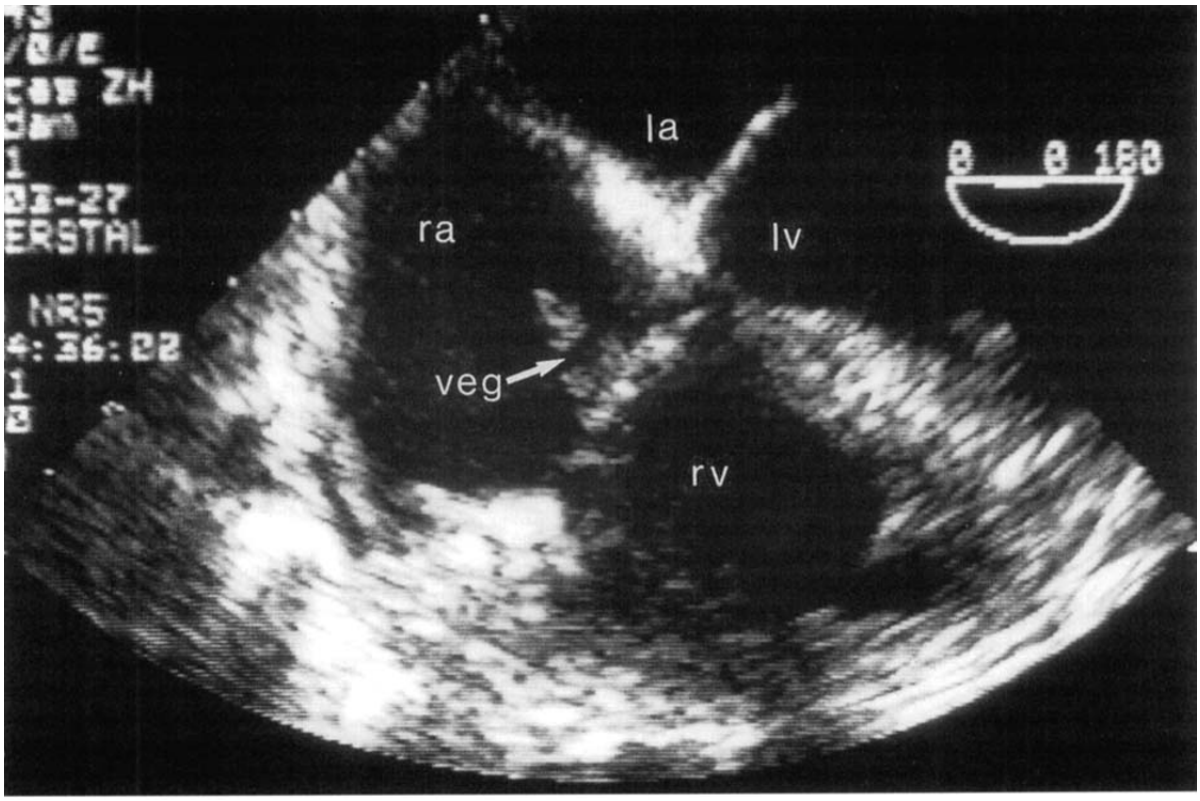

Figure 1 Transesophageal echocardiogram four-chamber view, showing a large tricuspid valve vegetation (indicated with arrow) prolapsing in systole into the right atrium. lA, left atrium; lv, left ventricle; ra, right atrium; rv, right ventricle.

regular pulse of 90/min and a body temperature of 37.6°C. The central venous pressure was elevated. Auscultation of the heart revealed no abnormalities. Breathing sounds were diminished with prolonged expiration. There was pitting edema of both legs.

Laboratory findings were as follows: ESR 5 mm/h, Hb 7.6 mmol/L, leukocytes 13.9×10^9/L with a normal differentiation. Chest X-ray showed enlarged pulmonary arteries without further abnormalities. The electrocardiogram showed sinus rhythm 80/min, with signs of right ventricular overload. The presumed diagnosis was right ventricular failure due to pulmonary hypertension in a patient with COPD. Treatment with diuretics was started. The day after admission she developed an intermittent fever up to 40°C with shivering attacks. She admitted to having had similar disturbances in the week before admission. Transthoracic echocardiography was performed, and showed right atrial and ventricular dilatation, tricuspid valve regurgitation with a calculated flow of 4.3 m/s and a floating mass on the anterior tricuspid valve leaflet. These findings were confirmed by a transesophageal echocardiogram (Figure 1). Because of the rapidly progressive right ventricular failure, the tricupid valve dysfunction, the floating mass and fever, a diagnosis of endocarditis was made.

Three blood cultures (three aerobic and three anaerobic bottles) were taken and empirical therapy with intravenous cefuroxime (3×1.5 g), intravenous flucloxacillin (4×1 g) and intravenous gentamicin (1×120 mg) was started. After an incubation period of 7 days one blood culture showed growth of a Gram-negative rod that seemed to be *C. canimorsus*. The identification of the organism was confirmed at the National Institute of Public Health and the Environment. The strain did not produce beta-lactamase. The patient was treated with intravenous penicillin (6×2 g) for 6 weeks. There was no relapse of fever and no further increase in right ventricular failure. Two weeks after the end of antibiotic therapy, transesophageal ultrasound showed a significant decrease in the size of the floating mass on the tricuspid valve.

C. canimorsus is an infrequent cause of bacteremia. However, its frequency as a pathogen may be underestimated because the results of blood cultures can be hampered by the slow growth of the organism and the prophylactic use of penicillin after dog bites. In the present case, only one of six blood-culture bottles was

positive. In another case report of tricuspid valve endocarditis, 38 blood cultures were needed to find the organism [3]. Our patient had none of the risk factors for infection with *C. canimorsus*. There was no history of alcohol abuse or steroid consumption. Abdominal ultrasound showed a normal spleen and her blood film did not suggest a non-functional spleen. There was no history of a dog or cat bite.

In two reviews of the literature on *C. canimorsus* infections, only 10% of the patients had endocarditis [2,4]. Usually the aortic or mitral valve is infected. Only three cases of isolated tricuspid valve endocarditis have been reported. Two of them had other cardiac abnormalities (tricuspid valve myxoma and atrial septum secundam) and the third patient was an alcoholic [3,5,6]. Our patient had no risk factor for endocarditis in general, or for *C. canimorsus* infection in particular.

We conclude that *C. canimorsus* should be considered as a cause of right-sided endocarditis even in immunocompetent patients without a history of a dog bite.

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References

1. Westwell AJ, Kerr K, Spencer MB, Hutchinson DN. DF-2 infection. Br Med J 1989; 298: 116–17.
2. Pers C, Gahrn-Hansen B, Frederiksen W. *Capnocytophaga canimorsus* septicemia in Denmark, 1982–1995: review of 39 cases. J Infect Dis 1996; 23: 71–5.
3. Andersen HK, Pedersen M. Infective endocarditis with involvement of the tricuspid valve due to *Capnocytophaga canimorsus*. Eur J Clin Microbiol Infect Dis 1992; 11: 831–2.
4. Hicklin H, Verghese A, Alvarez S. Dysgonic fermenter 2 septicemia. Rev Infect Dis 1987; 9: 884–90.
5. Worthington M, Gleason T, Pandian NG, Daly B. Tricuspid valve myxoma infected with dysgonic fermenter-2. South Med J 1984; 77: 241–2.
6. Niefield S, Young EJ. Native valve endocarditis caused by dysgonic fermenter-2 bacilli. Am J Med Sci 1988; 296(1): 69–70.